Neurology Asia 2026; 31(2) : 665 – 672

The relationship between the presence of neuropathic pain and quality of life, oxidative stress, and inflammatory parameters in hemodialysis patients

[1]Turgut Kültür, [2]Hakkı Öztürk, [3]Hatice Ağır, Aydın Çifçi, [4]Aykut Hacıömeroğlu, [4]Artuner Varlıbaş, [5]Salim Neşelioğlu, [5]Özcan Erel

[1]Department of Physical Medicine and Rehabilitation, Faculty of Medicine, Kırıkkale University, Kırıkkale, Türkiye; [2]Infectious Diseases Epidemiologist and Dialysis Unit, Private Ankara Dialysis Center, Ankara, Türkiye; [3]Department of Physical Medicine and Rehabilitation, Private Koru Hospital, Ankara, Türkiye; [4]Department of Internal Medicine, Faculty of Medicine, Kırıkkale University, Kırıkkale, Türkiye; [5]Department of Medical Biochemistry, Faculty of Medicine, Ankara Yıldırım Beyazıt University, Ankara, Türkiye

Abstract

Background & Objective: Neuropathic pain (NP) is common in hemodialysis patients and severely reduces their quality of life. Diabetes, inflammation, and oxidative stress are factors implicated in the development of NP, regardless of dialysis status. This study explored links between neuropathic pain, oxidative stress, inflammation, and quality of life in hemodialysis patients. *Methods:* The study prospectively included 113 HD patients, grouped by Leeds Assessment of Neuropathic Symptoms and Signs (LANSS) score (NP ≥12, control: <12).The groups were compared in terms of oxidative stress (native thiol, total thiol, disulfide, and Ischemia-Modified Albumin (IMA) levels) and inflammatory parameters (Systemic Immune-inflammation Index (SII)). The groups were also compared in terms of the Charlson Comorbidity Index (CCI), quality of life (SF-36), and disease perception (B-IPQ). *Results:* The native thiol/total thiol ratio was significantly higher in the NP group; however, disulfide levels, disulfide/native thiol, and disulfide/total thiol ratios were higher in the control group ($p<0.05$). There was no difference between the groups in terms of other parameters. NP presence was associated with a significant decrease in quality of life in all SF-36 subscales ($p<0.05$). Thiol/disulfide parameters were identified as independent predictors.
Conclusion: The presence of NP in hemodialysis patients is associated with impaired thiol/disulfide homeostasis, independent of diabetes and inflammation. Oxidative stress may play a dominant role in NP pathogenesis and that thiol/disulfide balance is a more sensitive biomarker compared to IMA.

Keywords: Hemodialysis, neuropathic pain, oxidative stress, thiol/disulfide homeostasis, ischemia-modified albumin.

INTRODUCTION

Neuropathic pain (NP) is a chronic pain syndrome characterized by hyperalgesia, allodynia, or spontaneous pain, arising from structural or functional abnormalities in the somatosensory system, and significantly impairing quality of life. Neuropathy is common in patients undergoing hemodialysis treatment due to chronic renal failure. Diabetes, inflammation, and oxidative stress are factors implicated in the development of NP, whether or not the patient is on dialysis.[1-3]

Reactive oxygen species (ROS) are natural byproducts of cellular metabolism. Under normal conditions, they are balanced by antioxidant defense systems; however, when they are overproduced due to hyperglycemia, obesity, or chronic diseases, this balance is disrupted and oxidative stress occurs. Oxidative stress plays a critical role in the progression of chronic diseases by causing damage to macromolecular structures.[4]

Address correspondence to: Turgut Kültür, 1Department of Physical Medicine and Rehabilitation, Kırıkkale University Faculty of Medicine, Kirikkale, Türkiye. Email: kurgut@ Hotmail.com

Date of Submission: 31 October 2025; Date of Acceptance: 12 January 2026

https://doi.org/10.54029/2026zey

Thiols are compounds containing sulfhydryl groups that play an important role in total antioxidant capacity. Their interaction with ROS results in the formation of reversible disulfide bonds, thereby maintaining thiol/disulfide homeostasis. This dynamic equilibrium is critical not only for antioxidant defense but also for fundamental biological processes such as apoptosis and cellular signaling. Disruption of thiol/disulfide homeostasis has been shown to play a role in the pathogenesis of many chronic diseases, including diabetes, cardiovascular disease, chronic kidney disease, and cancer. Therefore, assessing thiol/disulfide balance may provide valuable biomarkers for the diagnosis and prognosis of diseases.[5]

Another parameter associated with oxidative stress, ischemia-modified albumin (IMA), arises as a result of a decrease in albumin's metal-binding capacity. IMA has been proposed as a potential biomarker because it rises early in conditions such as myocardial ischemia and renal failure; however, its low specificity limits its use.[6]

The current literature has demonstrated that oxidative stress is closely related to a decrease in quality of life. Neuropathic pain also significantly negatively affects quality of life, and low quality of life has been reported to be associated with an increased risk of mortality.[7-9]

In this context, it is conceivable that the presence of NP in patients undergoing hemodialysis treatment for chronic renal failure may contribute to the progression of kidney disease by increasing oxidative stress.

This study was designed to evaluate the relationship between NP and oxidative stress in patients undergoing hemodialysis treatment due to chronic kidney failure. To this end, thiol/disulfide homeostasis and ischemia-modified albumin levels were examined; furthermore, the relationship of these biomarkers with neuropathic pain, their impact on comorbid diseases, inflammatory parameters, and quality of life were investigated.

METHODS

The study included a total of 113 patients aged 18 years and older who had been undergoing hemodialysis treatment for at least three months due to chronic kidney failure and who agreed to participate in the study. Participants were divided into two groups based on their Leeds Assessment of Neuropathic Symptoms and Signs (LANSS) score: the neuropathic pain group (LANSS ≥12) and the control group (LANSS <12).[10] Individuals who were not undergoing hemodialysis treatment, had a history of pregnancy, had a nervous system disease, or did not sign the consent form were excluded from the study. The Turkish validity and reliability study of the LANSS pain scale used for the diagnosis of neuropathic pain was conducted by Yücel *et al*.[10]

Blood samples were obtained from the residual portion of venous blood collected during routine hemodialysis sessions. Serum samples were separated after centrifugation and stored in sterile Eppendorf tubes at -80 °C until analysis. Participants' age, gender, hemodialysis duration, dialysis adequacy (Kt/V), concomitant chronic diseases, regular medication use, and laboratory parameters were recorded from patient files.

Thiol/disulfide homeostasis in biochemical analyses was measured using a fully automated spectrophotometric method developed by Erel and Neşelioğlu.[11] With this method, free thiol, total thiol, and disulfide levels were calculated to obtain thiol/disulfide ratios. The level of IMA was determined using a spectrophotometric method based on the decrease in albumin's cobalt-binding capacity.[12] The results of the biochemical tests were obtained from routine current examinations.

Complete blood count parameters (leukocytes, hemoglobin, platelets, mean platelet volume, platelet count) and biochemical tests (CRP, electrolytes, lipid profile, albumin, ferritin, parathyroid hormone, and ALT) were evaluated in all patients using the same device.

The systemic immune-inflammation index (SII) was calculated using the formula [neutrophils × platelets / lymphocytes] and used as a marker of inflammatory burden.[13]

Platelecrit (PCT) is a marker derived from the combination of PLT and MPV and calculated as PCT = (MPV x platelet count)/10000. This calculation provides more precise information than other platelet indices and offers a comprehensive assessment of total platelet mass. It has been proven to be an effective screening tool for detecting quantitative abnormalities in platelets. The PCT index has been used to assess the predictive value of prognosis in various medical conditions, including saphenous vein graft disease, polycythemia vera, liveoid vasculopathy, hepatitis A infection, and non-small cell lung cancer. Additionally, lower PCT levels have been found to be closely associated

with impaired peripheral nerve conduction function and the presence of neuropathy in type 2 diabetics. This correlation suggests that PCT may serve as a potential biomarker for distal symmetric polyneuropathy.[14]

The Charlson Comorbidity Index (CCI) was calculated for each participant to predict mortality risk.[15] Quality of life was assessed using the Short Form-36 (SF-36) questionnaire, and illness perception was assessed using the Brief Illness Perception Questionnaire (B-IPQ). Validity and reliability studies have been conducted for both scales in the Turkish population.[16,17]

The case report form was completed for all cases by carefully obtaining data from patient records, respecting patient confidentiality, including thiol/disulfide levels, age and gender data, CBC (complete blood count), CRP, urea, creatinine, biochemical data, quality of life tests, hemodialysis duration, regimen, duration of chronic kidney disease and underlying cause, additional chronic diseases, regular medication use, presence of neuropathic complaints, calcium binder use, phosphate binder use.

Statistical analysis

Statistical analyses were performed using SPSS 16.0 software. Continuous variables were presented as mean ± standard deviation or median (IQR), while categorical variables were presented as frequency and percentage. For intergroup comparisons, Student's t-test was used for variables showing a normal distribution, The Mann-Whitney U test for non-normally distributed variables, and the Chi-square test for categorical data. In the correlational analyses, Spearman's correlation was used to examine the associations between oxidative stress parameters and LANSS, SF-36, and B-IPQ scores. In univariate logistic regression analysis, each biomarker was included in the model individually, and odds ratios (OR) and 95% confidence intervals were calculated. Variables found to be significant in univariate analyses were included in the multivariate model, and independent predictors were determined. A p-value < 0.05 was considered the threshold for statistical significance.

RESULTS

A total of 113 chronic hemodialysis patients were included in the study. Thirty-seven participants formed the NP Group, while 76 formed the Control Group. There were no significant differences between the two groups in terms of age, gender distribution, body mass index, and duration of chronic kidney disease (Table 1). Similarities were also observed in terms of underlying comorbidities (diabetes mellitus, hypertension, coronary artery disease, congestive heart failure, chronic obstructive pulmonary disease, and malignancy).

Additional comorbidities and clinical histories were examined, there were no significant differences between the two groups in terms of parameters such as familial Mediterranean fever,

Table 1: Demographic and clinical baseline characteristics of Case (neuropathic pain) and Control Groups

Variable	Case Group (n=37)	Control Group (n=76)	p-value
Age (years)	67.05 ± 9.10	63.61 ± 8.56	0.06
Body mass index (kg/m²)	25.34 ± 5.53	26.11 ± 3.50	0.44
Male gender (n, %)	19 (51.4%)	45 (59.2%)	0.56
Duration of chronic kidney disease (years)	9.51 ± 10.58	8.42 ± 8.13	0.58
Dialysis adequacy (Kt/V)	1.55 ± 0.34	1.52 ± 0.29	0.62
Charlson comorbidity index ≥3 (n, %)	10 (27.0%)	17 (22.4%)	0.76
Diabetes mellitus (n, %)	12 (32.4%)	37 (48.7%)	0.15
Hypertension (n, %)	27 (73.0%)	45 (59.2%)	0.22
Chronic obstructive pulmonary disease (n, %)	4 (10.8%)	5 (6.6%)	0.68
Congestive heart failure (n, %)	5 (13.5%)	8 (10.5%)	0.88
Coronary artery disease (n, %)	6 (16.2%)	4 (5.3%)	0.12
Malignancy (n, %)	1 (2.7%)	1 (1.3%)	1.00

Table 2: Additional comorbidities and clinical histories of Case and Control Groups

Variable	Case Group (n=37)	Control Group (n=76)	p-value
Familial Mediterranean fever (n, %)	1 (2.7%)	1 (1.3%)	1.00
Renal biopsy (n, %)	2 (5.4%)	2 (2.6%)	0.84
Kidney transplantation (n, %)	0 (0.0%)	0 (0.0%)	–
Blood transfusion (n, %)	16 (43.2%)	34 (44.7%)	1.00
Peritoneal dialysis (n, %)	0 (0.0%)	1 (1.3%)	1.00
Antibiotic use in the last 6 months (n, %)	4(10.8)	7 (9.2)	0.78
Phosphorus binder intake (n, %)	8(21.6%/)	8(10.5%/)	0.11
Iron IV intake (n, %)	5 (13.5%)	4 (5.3%)	0.13
Calcitriol intake (n, %)	6(83.8%)	11(85.5%/)	0.80
Calcium binder intake (n, %)	17(45.9%)	34((44.7%/)	0.90

renal biopsy, and peritoneal dialysis history. Antibiotic use in the last 6 months, phosphate binder intake, iron IV intake, calcitriol intake, calcium binder intake, blood transfusion and frequency were also similar (Table 2).

In the comparison of laboratory parameters, serum disulfide levels were significantly higher in the Control Group (20.54 ± 3.88 µmol/L vs. 18.93 ± 3.27 µmol/L, p=0.02). Additionally, the disulfide/native thiol (%) and disulfide/total thiol (%) ratios were higher in the Control Group, while the native thiol/total thiol ratio was significantly higher in the NP Group ($p<0.01$ in all comparisons). No significant differences were found between the two groups in terms of other biochemical and hematological parameters, IMA level, Systemic Immune-inflammation Index (SII), and PCT (Table 3).

In the quality of life assessment, all subscales of the SF-36 were significantly higher in the control group compared to the neuropathic pain group ($p<0.01$). Particularly, significant differences were observed in physical role limitation, emotional role limitation, pain, social functioning, and general health scores. Disease perception (B-IPQ) was higher in the neuropathic pain group, indicating a more negative perception, but it showed borderline statistical significance (p=0.05) (Table 4).

In correlation analyses, disulfide ratios were found to have a positive correlation with SF-36 physical role limitation, while the native thiol/total thiol ratio showed a negative correlation. Additionally, IMA level showed a negative relationship with the SF-36 health change subscale (all correlations $r\approx0.20$, $p<0.05$) (Table 5).

In logistic regression analysis, disulfide, disulfide/total thiol ratio, and native thiol/total thiol ratio showed a significant association with neuropathic pain in univariate evaluation. In multivariate analysis, the disulfide/total thiol ratio and native thiol/total thiol ratio emerged as independent predictors ($p\approx0.045$). IMA, SII, and PCT were found to be non-independent predictors (Table 6).

DISCUSSION

This study examined the relationship between NP and oxidative stress in patients undergoing hemodialysis treatment due to chronic kidney failure. Our findings indicate that the presence of NP significantly reduces quality of life, that disruption in thiol/disulfide homeostasis may be associated with this clinical picture, and that specifically, the native thiol/total thiol and disulfide/total thiol ratios may be independent predictors. Our most striking finding was that the native thiol/total thiol ratio was significantly higher in patients with NP, whereas disulfide levels and disulfide-based ratios were found to be higher in the control group. This situation suggests that NP may be associated with a specific shift in the thiol/disulfide balance and that redox adaptation mechanisms may play a role in its pathophysiology.

One of the key findings of our study is that there were no significant differences between the groups in terms of diabetes, SII, and CRP levels, a common marker of inflammation. However, the literature emphasizes that diabetes and inflammation are major determinants of neuropathic pain and chronic pain syndromes

Table 3: Laboratory and oxidative stress parameters of Case (neuropathic pain) and Control Groups

Variable	Case Group (n=37)	Control Group (n=76)	p-value
Leukocytes (10^3/μL)	6.68 ± 2.89	6.94 ± 2.30	0.63
Neutrophils (10^3/μL)	4.76 ± 2.59	4.59 ± 1.94	0.72
Lymphocytes (10^3/μL)	1.25 ± 0.50	1.40 ± 0.51	0.12
Monocytes (10^3/μL)	0.61 ± 0.24	2.42 ± 11.20	0.16
Platelets (10^3/μL)	192.71 ± 92.50	198.99 ± 61.73	0.71
Platelecrit (%)	0.31 ± 0.25	0.29 ± 0.22	0.62
Hemoglobin (g/dL)	11.36 ± 1.92	11.63 ± 1.50	0.47
Hematocrit (%)	34.71 ± 8.31	35.20 ± 6.39	0.76
Mean platelet volume (fL)	11.17 ± 1.06	10.88 ± 1.07	0.19
HDL (mg/dL)	37.46 ± 8.58	40.13 ± 8.57	0.12
LDL (mg/dL)	82.11 ± 27.95	75.92 ± 27.23	0.27
Total cholesterol (mg/dL)	143.49 ± 36.32	149.53 ± 40.50	0.43
Triglyceride (mg/dL)	126.68 ± 62.94	155.92 ± 87.69	0.05
C-reactive protein (mg/L)	17.89 ± 28.03	18.62 ± 26.95	0.90
Ferritin (ng/mL)	703.03 ± 331.07	663.62 ± 316.12	0.55
Parathormone (pg/mL)	298.86 ± 274.99	301.91 ± 234.33	0.95
Sodium (mmol/L)	138.14 ± 3.36	138.00 ± 4.81	0.86
Potassium (mmol/L)	5.18 ± 0.80	5.37 ± 0.84	0.28
Calcium (mg/dL)	8.43 ± 0.92	8.57 ± 0.91	0.45
Phosphorus (mg/dL)	5.10 ± 1.40	5.33 ± 1.35	0.41
Alanine aminotransferase (U/L)	15.70 ± 17.26	14.93 ± 7.10	0.79
Albumin (g/dL)	3.65 ± 0.33	4.54 ± 4.90	0.12
Native thiol (μmol/L)	226.52 ± 39.34	232.47 ± 43.85	0.47
Total thiol (μmol/L)	264.38 ± 45.09	273.54 ± 50.45	0.33
Disulfide (μmol/L)	18.93 ± 3.27	20.54 ± 3.88	0.02
Disulfide / Native thiol (%)	8.39 ± 0.75	8.89 ± 1.04	<0.01
Disulfide / Total thiol (%)	7.18 ± 0.55	7.53 ± 0.74	<0.01
Native thiol / Total thiol (%)	85.65 ± 1.10	84.93 ± 1.48	<0.01
Ischemia-modified albumin (ABSU)	0.63 ± 0.19	0.67 ± 0.22	0.31
Systemic immune-inflammation index	854.36 ± 734.48	743.19 ± 480.98	0.41

Abbreviations: CRP, C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; PTH, parathormon; ALT, alanine aminotransferase; ABSU, absorbance unit; MPV, mean platelet volume.

in hemodialysis patients. Mizher and colleagues demonstrated a significant association between elevated CRP levels and chronic pain in hemodialysis patients, while Kallem *et al*. reported a link between high IL-6 and TNF-α levels and pain in diabetic neuropathy among end-stage renal disease patients.[18,19] However, the fact that no difference was detected in these parameters in our study indicates that neuropathic pain may arise directly from the effects of oxidative stress, independently of inflammation and diabetes. This finding suggests that oxidative stress during hemodialysis creates a unique pathophysiological burden and may be a primary factor in the development of neuropathic pain.

Oxidative stress plays a critical role in the pathogenesis of endothelial dysfunction, hypertension, atherosclerosis, and cardiovascular morbidity in hemodialysis patients.[20]

Table 4: Quality of Life and Illness Perception Scales of Case (Neuropathic Pain) and Control Groups

Variable	Case Group (n=37)	Control Group (n=76)	p-value
Illness perception (B-IPQ total score, 0–80)	54.78 ± 11.30	50.80 ± 6.48	0.052
SF-36 Physical functioning	47.57 ± 37.87	68.88 ± 25.83	0.003
SF-36 Physical role limitation	36.49 ± 43.14	73.36 ± 35.20	<0.001
SF-36 Emotional role limitation	36.94 ± 42.88	78.07 ± 36.34	<0.001
SF-36 Energy/vitality	45.14 ± 16.35	53.95 ± 11.29	0.005
SF-36 Mental health	53.84 ± 10.31	59.32 ± 9.76	0.009
SF-36 Social functioning	53.04 ± 28.63	76.15 ± 22.93	<0.001
SF-36 Pain	46.76 ± 34.30	74.14 ± 24.72	<0.001
SF-36 General health	33.65 ± 17.55	45.99 ± 10.65	<0.001
SF-36 Change in health	35.81 ± 21.68	49.67 ± 11.90	0.001

Abbreviations: B-IPQ, Brief Illness Perception Questionnaire; SF-36, Short Form-36

Furthermore, the accumulation of oxidative damage in nerve fibers in the pathophysiology of uremic neuropathy leads to structural and functional impairments, explaining the clinical findings.[21,22]

The thiol/disulfide homeostasis measurement used in our study is a dynamic and quantitative indicator of oxidative stress. Thiols are potent antioxidants containing sulfhydryl groups and neutralize ROS. Under oxidative conditions, thiols reversibly convert to disulfide bonds, and this conversion is one of the key determinants of cellular redox balance. A shift in the balance toward disulfides is indicative of increased oxidative stress.[4,5,11] The increase in the native tyrosine/total tyrosine ratio found in NP ground may reflect an adaptive antioxidant response. The different patterns observed in our findings suggest that oxidative stress in hemodialysis patients may have a specific phenotype associated with neuropathic pain. There was no difference between the two groups in terms of IMA levels. The literature also reports that IMA has low specificity and may increase in different clinical conditions.[6]

Our findings suggest that IMA may not be able to distinguish beyond the already high levels of oxidative stress present in dialysis patients, whereas thiol/disulfide parameters may emerge as more sensitive biomarkers. In the quality of life analysis, the presence of neuropathic pain was found to be associated with lower quality of life. This result is consistent with the literatüre.[8,23]

Phyo *et al.* have indicated that low quality of life is closely related to increased mortality.[9] In this context, the deterioration in quality of life observed in our study may be associated with an increased risk of mortality in patients undergoing hemodialysis who have NP. This issue should be investigated in further studies.

One of the strengths of our study is that it comprehensively evaluated not only the thiol/disulfide balance but also numerous clinical parameters.

Blood transfusion, type of vascular access, phosphate binder use, calcitriol intake, antibiotic use, renal biopsy, and additional chronic diseases were examined across a broad clinical spectrum; however, no significant association with neuropathic pain was identified. This

Table 5: Significant correlations between oxidative stress parameters and Quality of Life Scales

Oxidative Stress Parameter	Clinical Scale	Spearman r	p-value
Ischemia-modified albumin (ABSU)	SF-36 Change in health	–0.21	0.025
Disulfide / Total thiol (%)	SF-36 Physical role limitation	+0.20	0.029
Disulfide / Native thiol (%)	SF-36 Physical role limitation	+0.20	0.030
Native thiol / Total thiol (%)	SF-36 Physical role limitation	–0.20	0.031

Abbreviations: IMA, ischemia-modified albumin; SF-36, Short Form-36

Table 6: Logistic regression analysis of the relationship between oxidative stress parameters and neuropathic pain

Variable	Univariant OR (%95 CI)	p-value	Multivariate OR (%95 CI)	p-value
Disulfide (μmol/L)	0.89 (0.79 – 0.99)	**0.036**	–	–
Disulfide / Total thiol (%)	0.43 (0.22 – 0.84)	**0.013**	3.8×10^{57} ($9.2 – 1.6 \times 10^{114}$)	**0.046**
Native thiol / Total thiol (%)	1.52 (1.09 – 2.12)	**0.013**	9.5×10^{28} ($4.5 – 2.0 \times 10^{57}$)	**0.045**
Ischemia-modified albumin (ABSU)	0.39 (0.06 – 2.64)	0.340	0.52 (0.07 – 4.09)	0.531
Systemic immune-inflammation index	1.00 (0.99 – 1.00)	0.340	–	–
Platelecrit (%)	1.58 (0.29 – 8.71)	0.600	–	–

Abbreviations: OR, odds ratio; CI, confidence interval; IMA, ischemia-modified albumin; ABSU, absorbance unit.

finding supports the notion that oxidative stress biomarkers may be more directly related to neuropathic pain in this patient group.

Despite our findings, our study has some limitations. The limited sample size and the fact that it was conducted at a single center reduce the generalizability of the results.

Additionally, the question arises as to whether oxidative stress causes NP or whether NP increases oxidative stress. Although our regression analysis results suggest that oxidative stress is the primary triggering mechanism, larger-scale, multicenter, and prospective studies are needed to clarify the causal relationship.

In conclusion, this study demonstrates a significant association between NP and impaired thiol/disulfide homeostasis in hemodialysis patients. The fact that this association is independent of common risk factors such as diabetes, CRP, and inflammation suggests that oxidative stress may be an independent mechanism in the pathogenesis of hemodialysis-associated neuropathy. Based on our findings, therapeutic strategies targeting oxidative stress in the management of neuropathic pain, such as antioxidant treatments or dialysis modalities that improve redox balance, may contribute significantly to science. This topic should be investigated in further studies.

DISCLOSURE

Ethics: This study was planned using a prospective cross-sectional descriptive design and conducted in accordance with the principles of the Declaration of Helsinki. Approval for the research was obtained from the Kırıkkale University Non-Interventional Research Ethics Committee on 05/06/2024, numbered 2024.06.13. Written informed consent forms were obtained from all participants included in the study. All participants provided their written informed consent at the time of their initial assessment.

Data availability: The datasets used and/or analyzed in this study are available from the corresponding author upon reasonable request.

Financial support: None

Conflict of interest: None

REFERENCES

1. Jiang M, Wang Y, Wang J, Feng S, Wang X. The etiological roles of miRNAs, lncRNAs, and circRNAs in neuropathic pain: a narrative review. *J Clin Lab Anal* 2022;36:e24592. doi:10.1002/jcla.24592
2. Kitala-Tańska K, Kania-Zimnicka E, Tański D, Kwella N, Stompór T, Stompór M. Prevalence and management of chronic pain, including neuropathic pain, in dialysis patients with end-stage renal disease. *Med Sci Monit* 2024;30:e943808-1. doi:10.12659/MSM.943808
3. Pop-Busui R, Roberts L, Pennathur S, Kretzler M, Brosius FC, Feldman EL. The management of diabetic neuropathy in CKD and dialysis patients. *Am J Kidney Dis* 2009;55:365. doi:10.1053/j.ajkd.2008.10.050
4. Reddy VP. Oxidative stress in health and disease. *Biomedicines* 2023;11:2925. doi:10.3390/biomedicines11112925
5. Söğüt İ, Şenat A, İlhan AO, Sezen A, Erel Ö. Evaluation of thiol/disulfide homeostasis and other oxidative stress markers in patients undergoing hemodialysis. *Turk J Nephrol* 2021;30:17–24. doi:10.5152/turkjnephrol.2021.4396
6. Shevtsova A, Gordiienko I, Tkachenko V, Ushakova

G. Ischemia-modified albumin: origins and clinical implications. *Dis Markers* 2021;2021:9945424. doi:10.1155/2021/9945424
7. Zivkovic A, Jotic A, Dozic I, *et al*. Role of oxidative stress and inflammation in postoperative complications and quality of life after laryngeal cancer surgery. *Cells* 2024;13:1951. doi:10.3390/cells13231951
8. Mitsikostas DD, Moka E, Orrillo E, *et al.* Neuropathic pain in neurologic disorders: a narrative review. *Cureus* 2022;14:e22258. doi:10.7759/cureus.22258
9. Phyo AZZ, Freak-Poli R, Craig H, *et al*. Quality of life and mortality in the general population: a systematic review and meta-analysis. *BMC Public Health* 2020;20:1596. doi:10.1186/s12889-020-09777-z
10. Yücel A, Şenocak M, Kocasoy Orhan E, Çimen A, Ertaş M. Results of the Leeds assessment of neuropathic symptoms and signs pain scale in Turkey: a validation study. *J Pain* 2004;5:427–32. doi:10.1016/j.jpain.2004.07.001
11. Erel Ö, Neşelioğlu S. A novel and automated assay for thiol/disulphide homeostasis. *Clin Biochem* 2014;47:326–32. doi:10.1016/j.clinbiochem.2014.09.026
12. Bar-Or D, Curtis G, Rao N, E Lau. Characterization of the Co(2+) and Ni(2+) binding amino-acid residues of the N-terminus of human albumin. *Eur J Biochem* 2001;268:42–8. doi:10.1046/j.1432-1327.2001.01867.x
13. Hu B, Yang XR, Xu Y, *et al.* Systemic immune-inflammation index predicts prognosis of patients after curative resection for hepatocellular carcinoma. *Clin Cancer Res* 2014;20:6212-22. doi:10.1158/1078-0432.CCR-14-0442
14. Wei S, Pan X, Xiao Y, Chen R, Wei J. The unique association between the level of plateletcrit and the prevalence of diabetic kidney disease: a cross-sectional study. *Front Endocrinol* 2024;15:1345293. doi:10.3389/fendo.2024.1345293
15. Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chronic Dis* 1987;40:373-83. doi:10.1016/0021-9681(87)90171-8
16. Koçyiğit H, Aydemir Ö, Fişek G, Ölmez N. Kısa Form-36 (KF 36)'nın Türkçe versiyonunun güvenilirliği ve geçerliliği [Reliability and validity of the Turkish version of the Short Form-36 (SF-36)]. İlaç *ve Tedavi Dergisi* 1999;12:102–6. (Turkish)
17. Armay Z, Özkan M, Kocaman N, Özkan S. Hastalık algısı ölçeğinin kanser hastalarında Türkçe geçerlik ve güvenirlik çalışması [Turkish validity and reliability study of the illness perception scale in cancer patients]. *Klinik Psikiyatri* 2007;10:192–200. (Turkish)
18. Mizher A, Hammoudi H, Hamed F, *et al*. Prevalence of chronic pain in hemodialysis patients and its correlation with C-reactive protein: A cross-sectional study. *Sci Rep* 2023;13:5293. doi:10.1038/s41598-023-32442-2
19. Kallem CJ, Alghwiri AA, Yabes JG, *et al.* Association of symptoms and collaborative care intervention (TĀCcare) with systemic inflammation biomarkers in end-stage kidney disease. *Kidney360* 2024;5:1299-310. doi: 10.34067/KID.0000000000000512.
20. Higashi Y. Roles of oxidative stress and inflammation in vascular endothelial dysfunction-related disease. *Antioxidants* 2022;11:1958. doi:10.3390/antiox11101958
21. Hamed SA. Neurologic conditions and disorders of uremic syndrome of chronic kidney disease: presentations, causes, and treatment strategies. *Expert Rev Clin Pharmacol* 2019;12:61–90. doi:10.1080/17512433.2018.155299422.
22. Arekapudi A, Smith DI. Uremic neuropathy. In: Pathogenesis of Neuropathic Pain: Diagnosis and Treatment. Cham (Switzerland): Springer 2022 189-211.
23. Dos Santos PR, Mendonça CR, Hernandes JC, *et al*. Pain in patients with chronic kidney disease undergoing hemodialysis: A systematic review. *Pain Manag Nurs* 2021;22:605-15. doi: 10.1016/j.pmn.2021.03.004